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NOTES

Two Fatal Cases of Type E Adult Food-Borne Botulism with Early Symptoms and Terminal Neurologic Signs

HEMANTH BADHEY,[1] DENNIS J. CLERI,[1,2]* RICHARD F. D'AMATO,[1,2] JOHN R. VERNALEO,[1] VICKY VEINNI,[1] JONATHAN TESSLER,[1] ANDREW A. WALLMAN,[1] ANTHONY J. MASTELLONE,[1] MATTEO GIULIANI,[1] AND LISA HOCHSTEIN[2]

Department of Medicine[1] and Department of Pathology, Microbiology Division,[2] The Catholic Medical Center of Brooklyn and Queens, Jamaica, New York 11432

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Type E botulism, one of the least common forms of botulinal intoxication on the East Coast of the United States, is described for two elderly patients with chronic underlying disease. Both patients consumed tainted kapchunka, a salted, ungutted whitefish. Gastrointestinal symptoms and signs were prominent, but neurologic complaints, although noted soon after the consumption of the fish in one patient, did not progress until late in the course of the patient's illness. One patient exhibited both urinary retention, which was reported mainly in one outbreak of type E botulism (M. G. Koenig, A. Spickard, M. A. Cardella, and D. E. Rogers, Medicine [Baltimore] 43:517–545, 1964), and muscular fasciculations, which have been rarely reported.

Food-borne botulism is often suspected when the constellation of common-source gastroenteritis is present in patients with symmetrical descending weakness or paralysis (15) and cranial nerve palsies (2, 9, 19). Gastrointestinal symptoms are not seen invariably (8, 9), and individual cases can be diagnosed when neurologic signs are present either early or prominently in otherwise healthy individuals (9). The diagnosis may be delayed when neurologic manifestations occur late in the course of the illness (2). Timely treatment with equine antitoxin can be problematic, because early botulism mimics many food-borne gastroenteritidies (3, 5, 6, 8, 9, 11). This is especially true of type E botulism intoxication, in which gastrointestinal symptoms may be prominent (2, 9, 12). We report two cases of fatal type E botulism in elderly patients with significant underlying diseases.

Patient 1 was a 64-year-old female who had a myocardial infarction in 1964 and was discovered to have chronic renal failure in 1980. She was admitted to this medical center in August 1985 with complaints of nausea, vomiting, and diaphoresis, which had begun the night before admission, shortly after she ate fish. She was taking digoxin, furosemide, and diltiazem daily. Her husband had eaten the same fish, experienced similar symptoms, and presented himself in the emergency room on the same day, but was discharged when his condition improved after symptomatic treatment. The emergency room physician examined both the patient and her husband and could elicit no other complaints. The patient's oral temperature was 36.8°C, her pulse was 72/min and regular, her blood pressure was 130/80 mm Hg (1 mm Hg = 133.3 Pa), and her respiratory rate was 16/min. She had a II/VI systolic murmur at the left sternal border, venous stasis, and 2+ pitting edema of the ankles and feet. Her hematocrit at the time of admission was 30.7%, and her leukocyte count was 10,900/mm^3, with a differential of 57% neutophils, 15% bands, and 28% lymphocytes. Her blood urea nitrogen was 137 mg/dl, her serum creatinine was 7.4 mg/dl, and her serum potassium was 6.1 mM. Her chest X ray revealed cardiomegaly, T-waves were inverted in leads I and AVL, and nonspecific ST-T changes were present on her electrocardiogram. The patient complained of abdominal pain 13.5 h after coming to the emergency room (we estimated that this was 31 h after consuming the fish) and became unresponsive, with a weak pulse and sluggishly reactive pupils. She was intubated, placed on a respirator, and treated with vasopressors. An aspiration pneumonia was diagnosed and treated with intravenous penicillin G. The patient underwent peritoneal dialysis but died 34 h after admission (51 to 52 h after consuming the fish). No autopsy or further testing was performed.

Patient 2 was a 63-year-old male, the husband of patient 1, who was hypertensive, suffered from chronic headaches, and had had a myocardial infarction in 1964. He was seen in the emergency room with his wife 1 day before his admission, with complaints of nausea and vomiting which had begun shortly after he ate fish and honey the previous evening. He had had nausea, vomiting accompanied by abdominal pain, blurred vision, headaches, and a choking sensation 3 days earlier, after having consumed the same fish and honey. His physical examination 1 day before admission was unremarkable. He was treated with two doses of intramuscular prochlorperazine and was discharged after his condition improved. He returned the next day with nausea, vomiting, abdominal pain, weakness, blurred vision, and difficulty in urinating.

At this time, a diagnosis of botulism was considered. Family members were instructed to submit the suspect foods for analysis. The temperature of the patient was 36.4°C orally, his pulse was 84/min and regular, his blood pressure was 118/84 mm Hg (1 mm Hg = 133.3 Pa), and his respiratory rate was 20/min. He had abdominal discomfort on palpation, but his bowel sounds were normal, and no re-

* Corresponding author.

bound tenderness was elicited. A neurologic examination in the emergency room was normal. A second examination by another physician revealed sluggishly reactive pupils, but no blurred vision, diplopia, or other abnormal findings. An examination by a third physician showed no abnormal neurologic findings. The patient's hematocrit was 47.2, and his leukocyte count was 8,700/mm^3, with a normal differential. His blood urea nitrogen was 30 mg/dl, and his serum creatinine was 1.4 mg/dl. Sinus bradycardia with nonspecific ST-T changes was seen on his electrocardiogram.

That evening, the patient had to sit up in bed to ease his difficult breathing. Arterial blood gases on room air were pH, 7.419; PCO_2, 32.9; and PO_2, 64.6. The patient was given oxygen by a 35% ventimask, and a repeat blood gas revealed a pH of 7.40, a PO_2 of 98, and a PCO_2 of 39.2. The patient had an episode of chills; therefore, two sets of aerobic and anaerobic blood cultures were drawn, which were subsequently found to be sterile. The following day he experienced chest pain without a change in his electrocardiogram pattern, shortness of breath while using the ventimask, and chest wall muscular fasciculations. A physical examination at that time found the patient to be alert and comfortable. His throat was congested and his lungs were clear, and no abnormal neurologic finding was noted except the fasciculations. Diazepam (5 mg) was administered slowly intravenously. The patient remained awake, complained of abdominal discomfort, and became cyanotic during an attempt at passing a nasogastric tube. He was intubated, but further efforts to resuscitate him failed, and he was pronounced dead 35.5 h after admission. A postmortem examination by the medical examiner revealed severe coronary artery disease. The patient's serum, in the mouse toxicity test, was neutralized by trivalent botulinum antitoxin, and type E botulinal toxin was found in the kapchunka (a salted, ungutted whitefish), found in the patient's home (7).

First reported in the United States in 1899, food-borne botulism is a rare disease. The true incidence of botulism is not known; single cases may go unrecognized (10). The incidence of botulism rose during the Depression, coinciding with the increasing practice of home preserving of food products (9, 10). The biology of the toxin has been described elsewhere (16).

Early gastrointestinal symptoms with transient diarrhea occur a few hours to 8 days after the ingestion of contaminated food (9, 12). The incubation period overlaps with many both more and less common causes of gastroenteritis (2, 5, 8–10). Of 108 outbreaks of suspected botulism investigated by the Center for Disease Control between 1964 and 1969, only 37 were proven to be caused by botulism (8).

Clinical botulism is characterized by neurologic abnormalities, especially in otherwise healthy individuals (9). The most common symptoms in botulism are dysphagia, dry mouth, diplopia, and dysarthria (8, 10). Cranial nerve abnormalities and symmetrical descending weakness make the diagnosis most likely (8, 15). Urinary retention has been reported in five of eight patients with type E botulism (12). These symptoms can be confused with those of cerebral vascular accident involving the basilar artery or its branches, atypical Guillain-Barré syndrome, myasthenia gravis, Eaton-Lambert syndrome, trichinosis, polyneuritis of diphtheria, chemical intoxications, tick paralysis, or psychiatric disorders (8, 9). Rarely will staphylococcal food poisoning present with neurologic symptoms resembling botulism (6).

The symptoms of patient 2 could be attributed to either congestive heart failure, diffusion abnormalities from chronic obstructive pulmonary disease, or pulmonary embolism, and his urinary retention could be attributed to hypertrophy of the middle lobe of the prostate. The patient's earliest neurological complaint, blurred vision, was not consistently present. His respiratory difficulties stabilized under close observation. Muscular fasciculation, though rarely mentioned, has been reported (18, 19).

Type E botulism may be particularly difficult to diagnose, because gastrointestinal signs may be more prominent than neurologic signs (2, 8, 9, 19). The methods for isolating *Clostridium botulinum* from suspect foods and clinical specimens have been described (1). It should be emphasized that although the isolation of *C. botulinum* and detection of toxin from suspect foods is epidemiologically useful, it is not diagnostic because of the ubiquity of *C. botulinum* spores in the environment. Indeed, many species of salt- and freshwater fish, including whitefish, are contaminated with type E toxin-producing *C. botulinum*. The spores are usually found in the fish intestine (2). The clinical diagnosis is established by demonstrating the presence of the toxin in serum, feces, gastric contents, or vomitus or by the recovery of *C. botulinum* from the patient's feces (1). Use of spore selection techniques (1) facilitates the isolation of *C. botulinum* from clinical and food specimens. Identification is accomplished by carbohydrate fermentation, indole production, milk digestion, gelatin hydrolysis, lecithinase and lipase production, spore location, and determination of principal metabolic products. *C. botulinum* is among the few lipase-producing clostridia encountered in clinical specimens. A toxin neutralization test is required for definitive identification. The assistance and suggestions of a reference laboratory must be solicited in all suspect cases of botulism. Treatment cannot wait for definitive laboratory diagnosis. Electromyographic changes occur early and can suggest the disease in both infants and adults (4, 13). When the risk involved in treatment approaches the risk of having the disease, our experience has led us to the conclusion that electromyography should be performed as an emergency procedure.

Although other therapies have been suggested (9), the conventional treatment of botulism is cathartic cleansing of the gastrointestinal tract and administration of penicillin G and antitoxin. Trivalent ABE antitoxin is administered when the type of botulism is unknown. Emetics should be used with caution; nasogastric suction may be preferable (8, 9). Antitoxin given early in the illness clearly reduces mortality. This was first shown for antitoxin to type E botulinal toxin (9) and has recently been proven for type A intoxication (8, 17). In that study, the mortality for all patients over 60 years of age was higher, but to a lesser degree than that observed if the antitoxin was administered. Although ABE antitoxin does have an overall adverse reaction rate between 9 and 17%, the incidence of anaphylaxis is as low as 1.9% (3, 9, 14). Skin testing with the equine product may not preclude an anaphylatic reaction (11).

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